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Comparison between caudal bupivacaine and rectal paracetamol for postoperative analgesia in paediatrics under-going infra-umbilical surgery

By
Dr. El Sadig Ahmed El Mustafa
M.B.B.S, (University of Khartoum)

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Supervisor
Dr. Kamal M. E. El Mubashar
Associate Professor
Department of Anaesthesia
Faculty of Medicine
University of Khartoum

Dedications

To my family for their support

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List of abbreviations

ASA	American Society of Anaesthesiologist
CNS	Central nervous system
CSF	Cerebrospinal fluid
GABA	Gamma amino-butyric acid
IM	Intra-muscular
ITFs	Inducible transcription factors
NMDA	N-methyl-D-aspartate
PAG	Periaqueductal gray matter
PDES	Pain and discomfort evaluation scale
S	Sacral vertebrae
TENS	Transcutaenous electrical nerve stimulation
WDR	Wide dynamic range

ABSTRACT

A prospective randomized study was performed on 100 Sudanese children presenting for elective infra umbilical surgical procedures under general anesthesia. All patients were of ASA grade I and II. Within the age group 1-12 years. The postoperative analgesic effect of two drugs bupivacaine and paracetamol was compared. Patients in the study were randomly allocated into two groups. 50 patients in each group. Patients in group A received caudal bupivacaine (0.5 ml/kg) (0.25%) of bupivacaine. Those in group B received 20 mg/kg paracetamol rectally. 18% of patients in group A needed postoperative analgesia in the first 24 hours, 44.4% of them received intra-muscular pethedine while 55.6% received oral paracetamol.

64% of patients in group B needed postoperative analgesia in the first 24 hours, 60.8% of them received intra-muscular pethedine while 39.2% of patients received oral paracetamol.

- Need for postoperative analgesia in the first 24 hours between the two groups is significantly different.
- There was significant difference in time of administration of postoperative analgesic between the two groups.

- There was no significant difference in the type of post-operative analgesic between the two groups.

In conclusion caudal bupivacaine is more effective and patients were satisfied with it more than rectal paracetamol.

ملخص الأطروحة

هذه دراسة أمامية تجريبية في معالجة الألم الذي يحدث بعد العملية الجراحيـة عنـد الأطفال وذلك باستخدام عقارين ومقارنة مدى فعاليتهما.

تم اختيار مائة طفل مريض عشوائياً للدراسة وهم يندرجون في عمليـات جراحيـة اختيارية وقد تم تقسيمهم إلى مجموعتين. خمسون مـريض فـي كـل مجموعـة. مرضـى المجموعة (أ) تم حقنهم بعقار البيوبفاكين فوق الأم الجافية (إبيدورال) فى المنطقة العجزية بعد إجراء التخدير العمومي بجرعة قدرها 0.5 ملجم/ كجم بتركيـز 0.25% للعقـار. مرضـى المجموعة (ب) تم وضع عقار باراسيتامول داخل الشرج بجرعة قدرها 20 ملجم/ كجم بعـد إجراء التخدير العمومي. 18% من مرضى المجموعة (أ) احتاجوا إلى مزيل ألم إضافي فـي خلال الأربع والعشرين ساعة الأولى بعد العملية، 44.4% منهم تم حقـنهم بعقـار البثـدين بالعضل و55.6% منهم أعطى عقار باراسيتامول بالفم.

46% من مرضى المجموعة (ب) احتاجوا إلى مزيل ألم إضافي في خـلال الأربـع والعشرين ساعة الأولى بعد العملية، 60.8% منهم تم حقنهم بعقار البثدين بالعـضل و39.2% منهم أعطى عقار باراسيتامول بالفم.

هناك فرق إحصائي في الحوجة إلى مزيل ألم في خلال الأربـع والعـشرين سـاعة الأولى بين المجموعتين. ليس هناك فرق إحصائي في نوع مزيل الألم الإضافي. وهناك فرق احصائي فى زمن حقنه بين المجموعتين.

خلاصة الدراسة أن حقن البيوبفاكين خارج الأم الجافية أكثر فعالية ورضى المرضى أكثر من وضع عقار الباراسيتامول في الشرج.

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INTRODUCTION

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage.[1] Provision of pain relief after an operation is essential in children to permit rapid discharge from hospital of a comfortable patient who is free of complications, with parents who are reassured of their child well-being in the immediate and late post-operative period.

After having proved that children perceive, respond to and remember pain similarly to adults, several analgesic techniques have been proposed aiming at protecting children against metabolic, haemodynamic and psychological changes caused by surgical procedures.

Post-operative pain differs from other types of pain in that it is transient and improves in short time. Degree, effect and duration vary greatly. Young and elderly who are emotionally stable show lesser responses.[2]

Postoperative pain is associated with many adverse effects: it reduces tidal volume, vital capacity and functional residual capacity. Sympathetic over activity induced by pain leads

to tachycardia, hypertension and increases cardiac work and myocardial oxygen demand.

Caudal analgesia is used successfully in the provision of pain relief in children, but occasionally it may result in adverse effects as a result of more extensive block than is necessary. Caudal block may relief early post-operative pain but in the later period, systemic analgesics may be needed.[3]

Rectal administration of drugs in children is safe and provides convenient route for drug absorption. Paracetamal is available in pediatric suppository formulation. It provides analgesia in post-operative period, in addition respiratory depressant effects of opioids are avoided.[4]

In this study two drugs bupivacaine and paracetamol were compared to identify the most suitable drug as far as patient satisfaction and safety are concerned.[4]

LITERATURE REVIEW

Pain is defined as an unpleasant sensory and emotional experience associated with acute tissue damage. Pain may be acute or chronic or a symptom of disease.

Acute pain from nociceptive stimulation is an important biological warning that something is wrong. Persistent pain may become chronic, imposing emotional, physical, economic and social stress. It is one of the most costy health care problems for the society.[5]

Anatomy of pain:

Structures that mediate our appreciation of, and response to pain are categorized into two groups: Those that deal with the response to pain as an unpleasant sensation, and sensory discriminative aspects of pain.[6]

The cortex: a host of connections link higher cortical structures with pain-centered nuclei in the thalamus and brainstem. The major cortical pathways are:

- Sensory cortex S I.
- Secondary sensory cortex S II.
- Anterior insula.

- Cingulate gyrus.

- All six layers of dorsal horn mainly I, II and V rex-laminae.

The thalamus: Several of its multiple nuclei are concerned with pain. The lateral nuclei deal with sensory discriminative aspects, the medial ones with affective pain (the internal medullary lamina bounds the dorsomedial nucleus laterally and separates it from the anterior nuclei. Within the internal medullarly lamina are the inter laminar nuclei, including centromedian and centrolateral nuclei).

The midbrain: Here most of circuitry is involved in affective pain with extensive connections to the reticular system of the brainstem:

- The peri-aqueductal grey matter.
- Deep layers of superior colliculus.
- The red nucleus.
- Pretectal nuclei (anterior and posterior).
- Nucleus of Darkschewitsch.
- Interstitial nucleus of Cajal.
- Intercolliculus nucleus cuniformis and Edinger-Westphal nucleus.

The pons: The most important pain related nucleus in the pons is the locus coeruleus. This is full of noradrenaline containing neurons, which projects to a variety of brainstem structures that modulate pain through pathways that descend to the spinal cord.

The medulla: This is also involved in motivational, affective aspects of pain. The nucleus gigantocellularis and related nuclei, the lateral reticular nucleus and a variety of other nuclei. The raphe nuclei in the descending pathways suppress pain.

Peripheral nerve sensitivity:

Tissue damage results in a drop in pH and release of chemicals e.g. histamine to which small myelinated fibers are sensitive, fibers respond and generated electrical impulses that travel along the nerve to the dorsal horn of spinal cord.

On entering the spinal cord the pain signals take two different pathways to the brain via neospinothalmic tracts and paleospinothalamic tracts.

Nerve fibers within the tract terminate mainly in lamina I (marginalis) of dorsal horn where they excite second order nervous of neospinothalamic tract. They give rise to long fibers that cross immediately to the opposite side, through the anterior comissure. They then pass to the brain in the anterio-lateral columns.

Pain physiology:

Pain originates at the level of the tissues in various nociceptors. These nociceptors transmit to the spinal cord and via two types of nerve fibers. Small myelinated A delta fibers transmits fast responses to the CNS. This, which facilitates appropriate responses for the patient, e.g. withdrawal of affected limb from noxious insult.[7] Small ummyelinated C-fibers transmits information slowly and produce delayed responses resulting in dull aching pain.

Persistent nociception recruits and amplifies signals, which are transmitted from spinal cord to the brain resulting in pain. Changes in the spinal cord include reduced threshold responses and expanded receptive fields, which participate in " wind up" response, which results in significant pain experience at a later time.[8]

Types of pain:

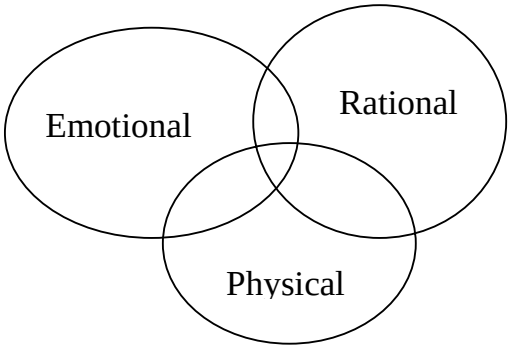
- ***Somatic pain:*** It is sharp, and well localized like pain arising from the skin, skeletal muscles and peritoneum, e.g. due surgical incision, second stage of labour pain and peritoneal irritation respectively.[9]
- ***Visceral pain:*** Pain receptors in the viscera are similar to those in the skin but are more sparsely distributed. Any event causing

stimulation of nerve endings in a viscus causes intense pain that is diffuse, poorly localized and associated with nausea and signs of autonomic nervous system activation. It radiates and causes referred pain at the same dermatomal origin as the affected viscus. Causes of viseral pain includes, ischaemia, ligamantal tears, smooth muscle spasms. Colicky type of visceral pain accompanies gastroenteritis, gall bladder and ureteral obstruction, menstruation, 1st stage of labour.[1]

Postoperative pain:

Postoperative pain is a complex physiologic reaction to tissue injury, visceral distention or disease. It results in an unpleasant, unwanted sensory and emotional experience. It is an extraoridinary complex sensation, which is difficult to define and measure. It may be defined as the sensory appreciation of afferent nociceptive stimulation, eliciting an effective autonomic component.[1]

The Ven diagram below shows the interrelationship between emotional rational and physical components of pain: the shaded area represents the quantum of suffering experienced by the patient.



Pain pathways:

a- Neural pathway:

First pain responses are conveyed from the periphery to the dorsal horn of the spinal cord in small myelinated fibers (A-delta). Second pain is conveyed in non myelinated (C) fibers.

b- ***Spinal cord pathways:***

Initial connections: 70% of fibers enter the dorsal root, the remaining enter the ventral (motor) root. Grey matter in the spinal cord has ten laminae. Most important in relation to pain are:

- Lamina I marginal zone.
- Lamina II substansia gelatinosa.
- Lamina V.
- Lamina VII & VIII intermediate spinal grey matter.

- Two main pathways of pain are:

1- Primitive spino-reticlo-diencephalic tract. Impulses pass from type C fibers to several second order neurons.

2- New neospinothalamic tract. Here most of the fibers are taken from lamina I & V, and mediate "first" pain.

Second order neurons:

1- Nerve cells that respond to gentle stimuli as well as pain, increasing their response as the stimulus increases. This is Wide Dynamic Range cell. WDR are found in lamina V. It has a wind up action, which occurs with repetitive stimulation via C fibers. Each added stimulus increases the response of WDR cell. It may be related to stimulation of glutamate receptors.

2- Nociceptive specific neurons found in lamina I responds to noxious stimuli.

3- Complex neurons (receives many inputs. Located in lamina VII and VIII.

Spinal pathways connections mediating "gating" are present here. Painful stimulation coming into the cord on C fibers can be modified by other inputs, which come from A delta fibers and B fibers.

This has practical consequences e.g.:

- Transcutaneous electrical nerve stimulation (TENS) works by high frequency, low amplitude stimulation of larger fibers, which inhibits transmission of pain through gates.
- Dorsal column stimulation.
- Making acupuncture effective.
- Rubbing skin locally to decrease pain.[10]

c- Higher ascending pathways:

i- Spino-reticulo diencephalic pathway (old): - It mainly ends in the reticular system of the brain stem as well as medial nuclei of thalamus. Emotional affective response to pain are due to projections that go from medial nuclei of thalamus to most of the cortex (anterior cingulate gyrus).

ii- Spinothalamic tract (new): connection here go to the sensory cortex (post central gyrus), but it is not the main pathway, since lesion along the pathway here do not cancel sensation of pain, but may cause severe pain due to possible damage of inhibitory pathway.[11]

d- Descending pathways:

Descending modulation of pain sensation originates from 3 main areas: the cortex, the thalamus and the brainstem. Fibers pass from PAG (periaqueductal greymatter) to the reticular

formation of the medulla (Ventromedulla) where connections are serotonergic and form axons descending in dorsolateral funicules of the spinal cord to end in interneurons next to substansia gelatinosa.

The synapses are encephalergic (lamina II) in the spinal cord. Stimulation of this system causes inhibition of incoming pain impulses therefore, serotonin applied peripherally augments pain. Its action centrally is important in descending inhibition of incoming painful impulses.[10]

Modulation of pain:

Pain in the periphery- the nociceptors:

Most tissues are provided with nociceptors. The quality of pain perceived on stimulation of nociceptors, depends on the site of stimulation and nature of fibers transmitting the sensation and type of stimulation in the periphery, there is a distinction between the sharp immediate pain transmitted by delta fibers and prolonged unpleasant burning pain mediated through unmyelinated C fibers.

Nociceptors have different receptors on the surfaces that modulate their sensitivity to stimulation, e.g. GABA, opiate, bradykinin, histamine, serotonin and capsicin receptors.

Nociceptors in the periphery lie dormant. Inflammation sensitizes a large number of nocieceptors, making then sensitive

to stimulation (hyperalgesia). Hyperalgesia may be primary (felt at the site of stimulation), related to sensitization of neurons innervating that area. It may be secondary (felt at a site remote from the original injury related to NMDA).[11]

Pain and neurotransmiters:

- Excitatory neurotransmitters: important are glutamate and tachykinins. These act at various neurokinin receptors including substance P, neurokinin A & B. Other substances that transmit pain impulses from incoming nerves in dorsal horn include calcitem gene related peptide, vasoactive intestinal polypeptide somatostatin and bombesin.
- Inhibitory neurotransmitters: in the central nervous system gamma amino-butyric acid (GABA) are the main inhibitory neurotransmitters.
- Descending pain regulation neurotransmitters: noradrenaline, alpha 2 stimulatory effects, serotonin and opiates relief pain by stimulating Mu and delta receptors at a host of sites.
- Specific neurotransmitters:

a- Glutamate: NMDA receptor mediates a host of spinal responses to severe stimulation. These receptors are inactive, due to Mg^{++} present on its ion channels to be remove, Mg^{++} adjacent peptide receptors have to be stimulated. Mg^{++} is

removed and painful stimuli occur. Glutamate receptor activation results in production of prostanoids and nitric oxide.

b- GABA: is spread in the brain and the spinal cord, a long with glycine. Interneurons in laminae I,II, III are GABA rich, and mediate gate control by synapsing on neurons that contain substance P. There are several distinct GABA receptors that work differently. The GABA A receptor is ligand gated ion channel. This allows chloride ions to leak into the cell, while GABA B receptor activates G proteins.

c- Tachykinins: neurokinin receptor mediate pain in the spinal cord. Substance P binds to NK-1 receptor, while neurokinins A & B bind respectively to NK-2 and NK-3 receptors. These substances are "tachykinins". The tachykinin receptors are G protein triggering gene transcription.[12]

Pain at the cellular level:

The cellular analogue of viral oncogene, and its cellular product, protein (fos); this is important in CNS changes that occur when we feel pain. Fos is one of the inducible transcription factors (ITFS) that controls mammalian gene expression. This could be a molecular marker for pain. C-fos can promote intracellular changes including cellular restructuring and proliferation. It is also involved in long term neurological consequences of noxious stimulation.

This noxious stimulation causes fos to appear in the spinal cord. Certain constitutive transcription factors change their activity.

Brief stimulation (10 min) causes ITFS to appear within 30 minutes, peak at one-two hours and disappears within 8 hours. Prolonged stimulation causes many fold increase in ITFS expression. Nociceptive C-fibers stimulation seems to be the main stimulus for ITFS production in the spinal cord.

Prolong stimulation causes C-fos to disappear from spinal neurons after 2-7 days. Production of ITFS leads to neuropeptide production and synthesis of a variety of receptors. C-Fos is involved in cell replication and differentiation.

Anaesthesia does not suppress production of c-fos within the spinal cord. Fentanyl reduces c-fos production by 50% and appropriate axial block with local anaesthetic agents can totally abolish c-fos response.[12]

Response to Pain:

Response to visceral pain is very different from somatic pain. Visceral pain results in tonic muscular spasm, while somatic pain usually causes withdrawal of affected part of the body, as protection from further damage.

Pain can have profound autonomic effects; there is crossover between the somatic and visceral systems at the level of

WDR cell in the spinal cord and extensively at higher centers, with projections to the hypothalamus.[11]

Since pain is a subjective and personal experience, its tolerance is described as a spectrum of individual experiences to the same or similar noxious event. In a study of human patients who can verbalize their level of discomfort, it was found that only 20% of patients felt that their pain experience was what they expected. It is therefore, impossible to predict pain levels.[13]

Adverse effects of pain:

Pain affects all systems:

- Respiratory system: in increased skeletal muscle tension hypoxaemia occurs. Decreased lung compliance results in hypercapnia and ventilation perfusion abnormality.
- Cardiovascular system: pain leads to increased myocardial work (mediated via catecholamines) causes dysarrhythmias, angina, myocardial infarction.
- Endocrine system: pain leads to increase in almost all hormones except insulin. Adenocorticotrophic hormone increases results in protein catabolism. Increased cortisol leads to lipolysis. Decreased insulin leads to hyperglycemia.
- Pain causes increased platelet adherence and activation of coagulation cascade, leading to increased incidence of

theromboemeoblic phenomena. In the gentiourinary system, pain increases sphincter tone and decreases smooth muscle tone leading to urine retention.[14]

Post-operative pain relief:

The commonest method of post-operative pain relief is the traditional use of on demand intra-muscular opioid injections. Postoperative pain is a common clinical problem with many adverse reactions.

Good post-operative management contributes to increased patient's confront as well as decreasing incidence of myocardial ischaemia, post-operative pulmonary complications and neuro-endocrine stress response. It also allows early mobilization of patients.[14]

Four classes of drugs are useful in management of postoperative pain.

1. Opioids e.g. morphine, pethedine and fentanyl.
2. Paracetemal and non-steroidal-anti-inflammatory drugs.
3. Nitric oxide: potent analgesic effective against somatic pain.
4. Local anaesthetics.

- **Non-pharmacological methods:**

- Cryotherapy: This may be applied to intercostal nerves exposed during a thoracotomy. Nerves are surrounded by ice-

ball produce sub-zero temperature at the end of the probe. Neural disruption is temporary and sensation returns after some months.[1]

- Transcutaneous electrical stimulation: (TES): a small alternating current is passed between two surface electrodes at low voltage, a frequency between 0.2 and 200 HZ. This increases CNS concentration of endorphines.[15]
- Acupuncture: it reduces pain and analgesic consumption especially after dental and abdominal surgery. It works in a similar manner as TES.[13]

• Caudal block: Analgesia for most surgical procedures of the lower part of the body (mainly below the umbilicus) can be provided by caudal block. The indications include herniotomy, operations of urinary tract, anus and rectum and orthopedic procedures on pelvic girdle and lower extremities.

- Complications are unusual: They result from misplacement of the needle into superficial soft tissues (failure of the block), intra-vascular or intra-osseous injections (systemic toxicity). Sub-arachnoid injection (spinal anaesthesia) or even penetration of pelvic viscera and vessels. These complications can easily be avoided by proper techniques.

Complication include:

1. Hypertension in patients more than 5 years old.
2. Delay in voiding, true urinary retention is rare.
3. Vomiting.
4. Needle trauma and intraneural injection result in pain.
5. Systemic toxicity following intravascular injection.

- ***Contra-indication:***

1. Infection at puncture site, septicemia, meningitis.
2. Bleeding disorders.
3. Allergy to local anaesthetics.
4. Un-corrected hypovolaemia.
5. Degenerative axon disease.

- ***Anatomy of the sacrum:***

The sacrum is a large triangular bone formed by the fusion of the five sacral vertebrae, articulating above with the fifth lumbar vertebra and below with the coccyx.

The posterior surface is convex and down its middle line runs the median sacral crest with is three or four rudimentary spinous processes. The laminae of the fifth and sometimes of the fourth sacral vertebrae fail to fuse in the midline; the deficiency thus formed is known as the sacral hiatus. The tubercles representing the inferior articular processes of the fifth sacral

vertebra are prolonged downwards as the sacral cornua. These cornua, with the rudimentary spine of the fourth vertebra above, bound the sacral hiatus. Four posterior sacral foramina correspond with the anterior foramina. Each transmits a sacral nerve posterior ramus and communicates with the sacral canal.

The apex is directed downwards and articulates with the coccyx. The coccyx represents four rudimentary vertebrae (sometimes three or five).

The sacral canal is a prismatic cavity running through the length of the bone and following it's curves from the lumber canal to the sacral hiatus (closed by the posterior sacrococcygeal membrane). Fibrous strands sometimes occur in the canal and divide the extradural space into compartments. These may account for some cases of failure to produce uniform analgesia. It's anterior wall is the sacral vertebrae; it's posterior wall, the laminae. Laterally four foramina are present. The anterior wall is sometimes very thin, easily pierced by a needle, which then enters marrow cavity. Aspiration reveals blood and injected drug rapidly enters the venous system.

The contents of the sacral canal are as follows:

1. The dural sac which ends at the upper border of the second sacral vertebra, on a line joining the posterior superior iliac spines. The pia mater is continued as the filum terminale.
2. The sacral nerves and the coccygeal nerve, with their dorsal root ganglia.
3. A venous plexus formed by the lower end of the internal vertebral plexus. These vessels are more numerous anteriorly than posteriorly and so the needlepoint should be kept as far posteriorly as possible.
4. Areolar and fatty tissue-more dense in males than in females.

The sacral hiatus is a triangular opening, caused by failure of the fifth (and sometimes of the fourth) laminar arch to fuse, with rounded apex formed by the fourth sacral spine, and a sacral cornua in each side below and laterally. It is covered over by the sacrococcygeal membrane, which is pierced by the coccygeal and fifth sacral nerves and filum terminale. It is superior to the sacrococcygeal junction, usually about 3.8 - 5 cm from the tip of the coccyx and directly beneath the upper limit of the intergluteal cleft.

Anatomical abnormalities of the sacrum are not uncommon. They include:

1. Upward or downward displacement of the hiatus;
2. Pronounced narrowing or partial obliteration of the sacral canal, making needle insertion difficult.
3. Ossification of the sacrococcgyeal membrane;
4. Absence of the bony posterior wall of the sacral canal, due to failure of laminae to fuse.
5. Dural extension to the level of S3-S4 in 2% of patients, quoted by Louis, or even to the sacrococcygeal membrane itself.
6. The hiatus may be of many different shapes, ranging from long and narrow to broad and shallow. The epidural space deep to it may range from being deep to excessively shallow. It may have a variable relationship to the tubercles.

The average capacity of the sacral canal is 34 ml in males and 32 ml in females. Its average length is 10-15 cm.

Technique:

A needle is inserted through the sacrococcygeal membrane at about 90 degrees to the skin surface in females and 45 degrees in males. Only after penetrating the membrane is the needle hub depressed towards the intergluteal cleft and the needle is advanced into the sacral canal. Moving the needle in this way

before piercing the membrane will lead the needlepoint into the subcutaneous tissues rather than the sacral canal. The point must not ascends higher than the line joining the posterior superior iliac spines and the dura, which ends at this level, be pierced. Occasionally (e.g. in children) the dural sac extends lower than S2. The mean distance between the apex of the hiatus and the dural sac is 4.5 cm.

After aspiration tests for blood and CSF have been proved negative, a test dose can be injected if thought necessary. If blood flow back through the needle, its tip is probably in the marrow cavity of the body of the vertebra, and must be re-sited correctly in the sacral canal. Should CSF appear, a decision must be made either to proceed to intradural injection, the proper amount of drug being introduced into the theca through the sacral needle, or to abandon the technique.

As a further test of entry to the sacral canal, 1 ml of air may be injected via the needle, while an assistant listens over the lumbar spine with a stethoscope. A whoosh of air is clearly heard via the stethoscope.

When the needle is correctly placed, injection is easy, no great force being required to depress the plunger of the syringe (except in patients with spinal stenosis). If the needle is

subcutaneous, injection of a few milliliters of air will produce surgical emphysema with it's crepitus, or a tumour is raised over the sacrum as the injection proceeds (only seen in thin patients). If the needlepoint comes to lie between periosteum and bone the force needed for injection will be great, a sure sign of an incorrect position. The dose is 0.5 -1 ml/kg of 0.25% bupivacaive.

Actions and clinical pharmacology of bupivacaine:

Local anesthetics block the generation and the conduction of nerve impulses, presumably by increasing the threshold for electrical excitation in the nerve, by slowing the propagation of the nerve impulse, and by reducing the rate of rise of the action potential. In general, the progression of anesthesia is related to the diameter, myelination, and conduction velocity of affected nerve fibers. Clinically, the order of loss of nerve function is as follows:

- Pain.
- Temperature.
- Touch.
- proprioception, and
- skeletal muscle tone.

Systemic absorption of local anesthetics produces effects on the cardiovascular and central nervous systems (CNS). At

blood concentrations achieved with normal therapeutic doses, changes in cardiac conduction, excitability, refractoriness, contractility, and peripheral vascular resistance are minimal. However, toxic blood concentrations depress cardiac conduction and excitability, which may lead to atrioventricular block, ventricular arrhythmias, and cardiac arrest, sometimes resulting in fatalities. In addition, myocardial contractility is depressed and peripheral vasodilation occurs, leading to decreased cardiac output and arterial blood pressure. Recent clinical reports and animal research suggest that these cardiovascular changes are more likely to occur after unintended intravascular injection of bupivacaine. Therefore, incremental dosing is necessary. Following systemic absorption, local anesthetics can produce central nervous system stimulation, depression, or both. Apparent central stimulation is manifested as restlessness, tremors and shivering progressing to convulsions, followed by depression and coma progressing ultimately to respiratory arrest. However, the local anesthetics have a primary depressant effect on the medulla and on higher centers. The depressed stage may occur without a prior excited state.

Pharmacokinetics:

The rate of systemic absorption of local anesthetics is dependent upon the total dose and concentration of drug administered, the route of administration, the vascularity of the administration site, and the presence or absence of epinephrine in the anesthetic solution. A dilute concentration of epinephrine (1:200,000 or 5 mcgm/mL) usually reduces the rate of absorption and peak plasma concentration of marcaine, permitting the use of moderately larger total doses and sometimes prolonging the duration of action. The onset of action with marcaine is rapid and analgesia is long lasting. The duration of analgesia is significantly longer with marcaine than with any other commonly used local anesthetic. It has also been noted that there is a period of analgesia that persists after the return of sensation, during which time the need for strong analgesics is reduced. The duration of anesthetic effect is prolonged by the addition of epinephrine 1:200,000. Local anesthetics are bound to plasma proteins in varying degrees. Generally, the lower the plasma concentration of drug the higher the percentage of drug bound to plasma proteins.

Depending upon the route of administration, local anesthetics are distributed to some extent to all body tissues, with high concentrations found in highly perfused organs such as the

liver, lungs, heart, and brain. Pharmacokinetic studies on the plasma profile of bupivacaine after direct intravenous injection suggest a three-compartment open model. The first compartment is represented by the rapid intravascular distribution of the drug. The second compartment represents the equilibration of the drug throughout the highly perfused organs such as the brain, myocardium, lungs, kidneys, and liver. The third compartment represents an equilibration of the drug with poorly perfused tissues, such as muscle and fat. The elimination of drug from tissue distribution depends largely upon the ability of binding sites in the circulation to carry it to the liver where it is metabolized.

After injection of bupivacaine for caudal, peak levels of bupivacaine in the blood are reached in 30 to 45 minutes, followed by a decline to insignificant levels during the next three to six hours. Various pharmacokinetic parameters of the local anaesthetics can be significantly altered by the presence of hepatic or renal disease, addition of epinephrine, factors affecting urinary pH, renal blood flow, the route of drug administration, and the age of the patient. The half-life of marcaine (bupivacaine) in adults is 2.7 hours and in neonates 8.1 hours.

Amide-type local anaesthetics such as marcaine are metabolized primarily in the liver via conjugation with glucuronic

acid. Patients with hepatic disease, especially those with severe hepatic disease, may be more susceptible to the potential toxicities of the amide-type local anaesthetics. Pipecoloxylidine is the major metabolite of marcaine.

The kidney is the main excretory organ for most local anesthetics and their metabolites. Urinary excretion is affected by urinary perfusion and factors affecting urinary pH. Only 6% of bupivacaine is excreted unchanged in the urine. When administered in recommended doses and concentrations, MARCAINE does not ordinarily produce irritation or tissue damage and does not cause methemoglobinemia.

Indications and usage:

Bupivacaine is indicated for the production of local or regional anesthesia or analgesia for surgery, diagnostic and therapeutic procedures. Only the 0.25% and 0.5% concentrations are indicated.

Bupivacaine hydrochloride:

Bupivacaine is a long acting, amide type local anesthetic chemically related to lignocaine and mepivacaine. It is approximately four times as potent as lignocaine.

Physical properties:

- Bupivacaine is presented in different concentrations. It is a clear, colourless, particle-free solution, with pH 4.0-6.5.
- Bupivacaine + Adrenaline is a clear, colourless, particle-free solution containing metabisulphite, with pH 3.3-5.0, metabisulphite may cause allergic-type reactions including anaphylactic symptoms and life-threatening or less severe asthmatic episodes in certain susceptible people.
- Bupivacaine should be stored at 25°C or below. Do not freeze.
- The ampoules are designed for single use only; any unused portions of solutions should be discarded.

Pharmacokinetics:

Bupivacaine has a pKa of 8.1 and a high degree of lipid solubility with an oil/water partition coefficient of *27.5*. It is mainly bound to alpha-1-acid glycoprotein in plasma with plasma binding of 96%. These factors contribute to its prolonged duration of action.

The rate of absorption and plasma concentration of bupivacaine depends upon the dose, the route of administration

and the vascularity of the injection site. Absorption may be slowed by the addition of adrenaline. In concentrations of 5 mg/ml, it has a long duration of action, from 2-5 hours following a single epidural injection and up to 12 hours after peripheral nerve blocks.

When used in low concentrations (2.5 mg/ml or less) there is less effect on motor nerve fibres and the duration of action is shorter. Low concentrations may, however, be used with advantage for prolonged pain relief, e.g. in labour or postoperatively. Absorption of bupivacaine from the epidural space occurs in 2 phases; the first phase is in the order of 7 minutes and the second is in 6 hours, the slow absorption is rate limiting in the elimination of bupivacaine, which explains why the apparent elimination half-life after epidural administration is longer than after intravenous administration. Bupivacaine has a total plasma clearance of 0.58 L/min, a volume of distribution at steady state of 73 L, an elimination half-life of 2.7 h and an intermediate hepatic extraction ratio of 0.4 following experimental IV administration in adults. The terminal elimination half-life is prolonged in the newborn to approximately 8 hours. In children over 3 months, the elimination half-life is similar to that in adults. Bupivacaine readily crosses the placenta and is excreted in breast milk in concentrations lower than the maternal plasma concentration.

Hepatic and renal disease, route of administration, age of the patient and certain concomitant medication can change the pharmacoketic parameters.

Metabolism of Bupivacaine:

Bupivacaine is metabolized in the liver and excreted via the kidneys, the possibility of bupivacaine accumulation should be considered in patients with hepatic and/or renal impairment. Bupivacaine is excreted in the urine principally as metabolites with about 6% as unchanged medicine and approximately 5% at the N-dealkylated metabolite, pipecolylxylidine (PPX). Following epidural administration, the urinary recovery of unchanged bupivacaine is about 0.2%, of pipecolylxylidine (PPX) about 1% and 4-hydroxy- bupivacaine about 0.1% of the administered dose.

Mode of Action:

Bupivacaine, like other local anesthetics, causes a reversible blockade of impulse propagation along nerve fibres by preventing the inward movement of sodium ions through the nerve membrane. Local anaesthetics of the amide type are thought to act within the sodium channels of the nerve membrane.

Given as a spinal anaesthetic, bupivacaine has a rapid onset and a medium to long duration. The duration is dose-

dependent. It is approximately four times more potent and toxic than lignocaine.

Indications:

- Analgesia in labour,
- Post-operative analgesia.
- Other therapeutic pain blocks, particularly where long-acting anaesthesia is required.
- Surgical anaesthesia.

Contraindications:

1. Allergy or hypersensitivity to amide type local anaesthetics or sodium metabisulphite in adrenaline-containing solutions.
2. Obstetric paracervical block, intravenous regional anaesthesia (Bier's block) and all intravenous infusions.
3. The following are additional contraindications for solutions with Adrenaline:

Adrenaline is contraindicated in conditions where the production or exacerbation of tachycardia could prove fatal such as:

- Thyotoxicosis.
- severe heart disease.

- in obstetrics when maternal blood pressure exceeds 140/90 mm Hg
- Adrenaline-containing solutions must not be used for analgesia in parts of the body with compromised blood supply or supplied by end arteries, such as fingers, toes, nose, ears or penis. There is a possibility of producing arterial vasoconstriction and subsequent ischaemic gangrene distal to the site of injection.

Side effects:

Cardiovascular: hypotension, bradycardia, arrhythmias and cardiac arrest may occur.

Respiratory: difficulty in breathing, apnoea and respiratory failure may be precipitated.

Central Nervous system: CNS manifestations are excitatory and/or depressant and may be characterized by light-headedness, tinnitus, nervousness, apprehension, euphoria, confusion, dizziness, drowsiness, blurred vision, vomiting, sensations of heat, cold or numbness, twitching, agitation, difficulty in swallowing, slurred speech, tremor, convulsions, unconsciousness.

Allergy: it may be presented as allergic der matitis, bronchospasm or anaphylaxis.

Acute systemic toxicity: It occurs in accidental intravascular injections and over dosage. The early features are circumoral paraesthesia, numbness of the tongue, light-headedness, hyperacusis and tinnitus, followed by cardiovascular and respiratory failure.

Precautions:

Caution in the presence of hepatic insufficiency, impaired cardiovascular function (severe bradycardia, cardiac conduction disturbances, severe shock and heart block), epilepsy and pre-existing abnormal neurological or neuromuscular disease. Reduction of the dosage in elderly, debilitated patients and in peadiatric patients.

Presentation:

Marcain ± Adrenaline

Bupivacaine hydrochloride with and without adrenaline.

- 0.125% infusion-a clear, colourless, particle-free solution containing. 1.25 mg/ml bupivacaine HCl, 8.5 mg/ml sodium chloride, with pH 4.0-6.5.

- 0.25% injection and infusion-a clear, colourless particle-free solution containing. 2.5 mg/ml bupivacaine HCl, 8 mg/ml sodium chloride, with pH 4.0-6.5.
- 0.37% injection -a clear, colourless particle-free solution containing. 3.75 mg/ml bupivacaine HCl, 8 mg/ml sodium chloride, with pH 4.0-6.5.
- 0.5% injection -a clear, colourless particle-free solution containing. 5 mg/ml bupivacaine HCl, 8 mg/ml sodium chloride, with pH 4.0-6.5.
- 0.25% injection plus adrenaline 1:400,000 -a clear, colourless particle-free solution containing 2.5 mg/ml bupivacaine HCl, 8 mg/ml sodium chloride, 4.5 mcg/ml adrenaline acid tartrate, 0.5 mg/ml sodium metabisulphite, with pH 3.3-5.0.
- 0.5% injection plus adrenaline 1:200,000 -a clear, colourless particle-free solution containing 5 mg/ml bupivacaine HCl, 8 mg/ml sodium chloride, 9.1 mcg/ml adrenaline acid tartrate, 0.5 mg/ml sodium metabisulphite, with pH 3.3-5.0.

Paracetamol:

Paracetamol is widely used in the management of pain and fever in children, having gained ascendancy after the reported association between Reye's syndrome and aspirin in the 1980s.

The drug is an effective antipyretic at plasma concentrations of 0.066-0.130mmol/l and it is assumed that analgesia occurs in a similar range. This is unproven. The analgesic effect of paracetamol is thought to be directly related to its plasma concentration, because of its high lipid solubility and low protein binding. Paracetamol is a weak acid with a high pKa and, in the alkaline medium of the duodenum, it is non-ionised and rapidly absorbed. Paracetamol absorption is used as a measure of gastric emptying. The hepatic extraction ratio is less than 0.3. Paracetamol is the most common drug prescribed in paediatric practice. In our own hospital, approximately 50% of inpatient children are prescribed this medication.[17]

Pain Relief from Paracetamol:

A direct relationship between plasma paracetamol levels and analgesia has been established in a rat model. Uric acid was injected into the knee joint of the hind limb, to act as nociceptive stimulus. The animals were then given paracetamol in doses of up to 562 mg/kg and recovery of function over time was considered as an expression of analgesia.

In adult human volunteer studies, paracetamol is superior to placebo as an analgesic. Both 0.5 g and 1g immediate release paracetamol were superior to placebo for one to five hours after

experimental pain induced by brief cutaneous application of argon laser pulses. The analgesic effect was assessed as a change in pricking pain threshold and no difference in analgesic effect was noted between these doses. In a further study, comparing paracetamol lg, paracetamol 1g plus codeine 60mg and placebo, pain threshold and brain evoked potentials to laser stimulation were assessed for six hours. The pain threshold was significantly elevated one and two hours after paracetamol ingestion. Paracetamol l g plus codeine 60 mg was superior to placebo for up to six hours after medication.

This view is supported by Rusy,[17] who demonstrated low or even undetectable serum paracetamol concentrations in the first 40 minutes after surgery, when rectal paracetamol 30-35 mg/kg had been administered intraoperatively. Mather[17] demonstrated a need to supplement rectal paracetamol 20 mg/kg with a nonsteroidal anti-inflammatory agent to achieve satisfactory analgesia. Adequate analgesia in children undergoing surgery has been described using preoperative oral paracetamol in a dose of 40 rng/kg. The cumulative frequency of children having satisfactory pain scores increased to a ceiling of 66% at a plasma paracetamol concentration of 0.25 mmol/l. Few additional children achieved analgesia with higher plasma concentrations.

Toxicity:

Hepatic toxicity is reported with plasma concentrations above 0.8 mmol/1 after acute poisoning. Concern about toxicity is the main reason for reticence among practitioners to prescribe higher than traditional doses of paracetamol. Paracetamol overdose results in increased production of highly reactive electrophilic arylating metabolites by the hepatic cytochrome P-450-dependent mixed function oxidase enzyme system. These metabolites bind to intracellular hepatic macromolecules to produce cell necrosis and damage. Paracetamol may accumulate in paediatric patients after repeated therapeutic doses. There is evidence, from adults, of glutathione depletion in volunteers given doses of 0.5 g and 3 g paracetamol separated by four to ten days. Penna and Buchanan20 reported seven deaths and 11 cases of hepatotoxicity associated with paracetamol poisoning in children. Mortality due to hepatotoxicity was associated with doses greater than 300 mg/kg/day for one to six days. Survival was usually seen in those children suffering hepatotoxicity due to paracetamol in doses greater than 150 mg/kg/day for two to eight days. Current guidelines recommend that doses should not exceed 90 mg/kg/day.

Infants:

Out of the neonatal period, the metabolism of paracetamol by infants is similar to older children (T1/2b 2.1, CI 0.365 l/kg/h). Recommended dosage regimes are conservative (60 mg/kg/day), again reflecting the lack of well conducted pharmacokinetic studies.

Lag times between concentration and effects:

Paracetamol is thought to have an analgesic effect via NMDA receptors in the spinal cord. Nielsen et al and Ariendt-Nielson *et al*[17] demonstrated a one hour delay between peak plasma paracetamol concentrations and maximum analgesia. Similarly, there is a 100 minute lag between peak plasma paracetamol concentration and peak temperature reduction. Cerebrospinal fluid concentrations of paracetamol mirror those in plasma with a similar time delay. It is thus prudent to administer paracetamol at least 1 hour orally or 2 hours rectally before a surgical insult.

The dose of paracetamol used in this study is 20 mg/kg, put in rectum in the lithotomy position after induction of anaesthesia.

Summary:

Paracetamol remains the stalwart of paediatric analgesia, despite limited evidence of efficacy. Concern about hepatotoxicity has resulted in cautious perioperative dosing regimes, but both pharmacokinetic and pharmacodynamic data have shown these doses to be inadequate. While there is increasing evidence that a single rectal loading dose of 20 mg/kg results in more desirable plasma paracetamol concentrations, caution must be taken not to exceed the current recommended daily dosing of 90 mg/kg/day.[17]

Previous studies:

A study done by TCK, and Roos showed that caudal block is the most commonly performed paediatric block for providing postoperative pain control for ambulatory surgery in children. Features of paediatric anatomy and physiology allow successful performance of the techniques.[18]

Another study done by Nielsen and Steels showed that regional analegesia in children provides a continuum of perioperative care that include peri-operative pain management, decreased opioid requirements, decreased postoperative nausea and vomiting. In addition regional analgesia has been shown to improve the cardiovascular, pulmonary, gastro-intestinal,

coagulative, immunological and cognitive functions. And to be of benefit of economic context.[19]

A study done by Wucl and Caldwell revealed thay the pathophysiology that commonly followed surgery result in detrimental physiological effects and may be associated with postoperative morbidity and mortality. The use of epidural analgesia but not systemic opioid may attenuate these effects and facilitate return of gastro-intestinal function, attenuate hyper-coagulable events and decrease postoperative pulmonary complications. And also facilitate patient recovery.[20]

In a double blind study done by Batra, Prasad, Arya, Chari and Yaddanapudi comparing caudal marcaine and tramadol in postoperative pain score and side effects. The result point towards a significantly lower pain score with marcaine and also vomiting is less frequent.[21]

A study of Dieng, Diouf and Diene showed that caudal marcaine is safe and secure procedure. Give pain relief even in painful procedures and good postoperative status with only some minor complications.[22]

A study done by Seymour showed that rectal paracetamol is an effective anagesic for controlling postoperative pain.[23]

OBJECTIVES

1. To assess the quality of analgesia when using caudal bupivacaine or rectal paracetamol.
2. Duration of analgesia of caudal bupivacaine and rectal paracetamol.
3. Side effects of caudal bupivacaine and rectal paracetamol.

PATIENTS AND METHODS

- **Study site:**

This study was performed in Soba university hospital in the period from 10th of March to 29th of September 2003. The availability of patient and the well-equipped theatre were the main reasons for selecting this hospital as study area.

The study has been approved by the ethical committee of the Faculty of Medicine and the patient's parents verbal consent were obtained.

- **Study population:**

A prospective study was performed on 100 patients presenting for different elective surgeries (herniotomies, hydroceles, vesical stones, un-descended testes, hypospadius, lower limb orthopedic surgery etc ...). The operations were done under general anesthesia using inhalation induction by halothane, suxamethonium for intubation. Maintenance by halothane and pancuronium together with oxygen and nitrous oxide, spontaneous or controlled ventilation.

- **Study design:**

The study utilized the experimental research design and the 100 patients included in the study were divided into two groups.

- ***Group A:*** consisted of 50 patients given caudal bupivacaine, dose 0.5 ml/kg of 0.25% bupivacaine.
- ***Group B:*** consisted of 50 patients given rectal paracetamol, dose 20 mg/kg.

- **Inclusion Criteria:**

1- Patients of ASA grade I and II.

2- Patients age between 1-12 years old.

3- Patients under going elective infra-umbilical surgery.

- **Exclusion Criteria:**

1- Patients of ASA grade III, IV, V.

2- Patients less than one year old and more than 12 years old.

3- Patients with neurological deficit in lower limbs.

4- Patients with malformation of the vertebral column.

5- Patients with sepsis at the site of caudal black.

6- Patients with bleeding disorders.

7- Patients with allergy to paracetamol or local anaesthetic.

- **General characteristics of patients:**

These were obtained from history and examination and were recorded. They include age, weight and blood pressure. All parents were asked about the past medical and anaesthetic history of their children (whether the patients had any complications following previous anaesthetic exposure). A full physical examination to all patients was conducted during pre-operative assessment and routine pre-operative investigations were observed.

- **Data collection:**

The data collected included the following parameters:

1. Drug used: caudal marcaine or rectal paracetamol.
2. Duration of analgesia.
3. Assessment of post-operative analgesia by pain and discomfort evaluation score (PDES) (appendix)
4. Need for pain killer.
5. Level of systolic blood pressure.
6. Complications.

All of the observations were recorded in the patient's charts. All drugs that were given to the patients throughout the operation and till recovery were recorded in the anaesthetic sheet.

Techniques:

- **Caudal bupivacaine:** after induction of general anaesthesia. Patient tilted to the left side, sacral hiatus identified at the midline by palpation, I washed my hands and wear gloves. The back washed with bovidone and spirit. Bupivacaine 0.5 ml/kg (0.25%) prepared in syringe, 21 gauge needle applied to the syringe and skin penetrated at 30 degree, cephalad direction at the sacral hiatus until I felt piercing sacrococcygeal membrane. Aspiration test was done if no blood or CSF obtained, then the drug injected slowly.
- **Rectal paracetamol:** after induction of general anaesthesia, the patient put in the lithotomy position, after washing hands and wear gloves. Paracetamol suppository was put in the rectum (20 mg/kg).
- Assessment of pain postoperative done every 15 min for one hour and then after 2 hours, 8 hours by anaesthesia registrar with collaboration with the surgical registrar on duty.
- The need for postoperative analgesia decided by both registrars according to the severity of pain.

RESULTS

This study included 100 patients divided equally into two groups according to the type of analgesia received. The groups were categorized into caudal bupivacaine and rectal paracetamol. The age of patients ranged from 1-12 years mostly from 2-4 years (**Table I**).

The male patients were 76% , 54% received caudal bupivacaine while 46% received rectal paracetamol. The female patients were 24%, 37% received caudal bupivacaine while 63% received rectal paracetamol. (**Table2**)

There were no statistical differences between the two groups regarding age, gender (P >0.05) (**Table II**) and duration of surgery (**Table III**).

Eighteen percent of patients who received caudal bupivacaine needed systemic analgesia in the first 24 hrs, 44.4% of them received I.M pethedine, while 55.6% received oral paracetamol (**Table 4**).

18% of patients who received caudal bupivacaine need supplementary analgesia, 44.4% received I.M pathedine while 55.6% received oral paracetamol.

46% of patints who received rectal paracetamol needed systemic analgesic, 60.8% of them received I.M pethedine . 39.2% received oral paracetamol. (**Table 5**)

33.3% of patients who received caudal bupivacaine need supplementary analgesia within the first 8 hours while 66.7% of them need after the first 8 hours.

36% of patients who received rectal paracetamol need supplementary analgesia within the first 8 hours, while 46% of them need after the 8 hours. (**Table 6**)

Comparing patients' satisfaction in relation to type of analgesia, there was significant difference between the two groups. Patients who received caudal bupivacaine were more satisfied than those who received rectal paracetamol.

The complications of caudal bupivacaine encountered by 16% of patients is pain at the site of injection. No complications were encountered related to rectal paracetamol. (**Table 7**)

The patients understudy had undergone various types of surgeries most lasting between 30-45 minutes (**Figure 1**). Manifestations noted after surgery were changes in systolic blood pressure, weeping, movements, anxiety, posture and pain complaint. Most groups showed no significant deviation from normal (**Figure 2**).

Pain was evaluated using pain and discomfort evaluation scores (PDES): Sixty percent of patients receiving caudal bupivacaine scored < 3. Twenty-six percent scored from 3 - 6 and 14% scored more than 6 (**Figure 3**).

60% of patients receiving caudal bupivacaine, undergoing urogenital surgery scored less than 3 while 26% of patients scored from 3- 6. 14% of patients scored from 6- 12. (**Figure 4**)

45.5% of patients receiving rectal paracetamol, undergoing urogenital sergery scored < 3. 28.6% of patients scored between 3- 6. 25% of patients scored between 6- 12. (**Figure 5**)

Table (1): Age (in years) distribution among the patients in the two groups:

group / Age (years)	Caudal bupivacaine	Rectal paracetamol	Total
< 2	8 (16%)	9 (18%)	17 (17%)
2 - 4	14 (28%)	17 (34%)	31 (31%)
4 - 6	9 (18%)	6 (12%)	15 (15%)
6 - 8	8 (16%)	7 (14%)	15 (15%)
8 - 10	6 (12%)	7 (14%)	13 (13%)
> 10	5 (10%)	4 (8%)	9 (9%)
Total	**50**	**50**	**100 (100%)**

Table (2): sex distribution among patients in the two groups.

group / Sex	Caudal bupivacaine	Rectal paracetamol	Total
Male	41(82%)	35 (70%)	76 (76%)
Female	9 (18%)	15 (30%)	24 (24%)
Total	**50**	**50**	**100 (100%)**

Table (3): Duration of surgery among two groups.

Group / Duration	Caudal bupivacaine	Rectal paracetamol	Total
< 15 min	3 6%	5 10%	8 8%
15 -30 min	14 28%	15 30%	28 29%
30 - 45 min	22 44%	19 38%	41 41%
45 - 60 min	7 14%	8 16%	15 15%
> 60 min	4 8%	3 6%	7 7%
Total	50	50	100

Table (4): Need of post-operative analgesia in the first 24 hours among the two groups.

Group / Need of pain killer	Caudal bupivacaine	Rectal paracetamol	Total
Yes	9 (18%)	23 (46%)	32
No	41 (82%)	27 (54%)	68
Total	**50**	**50**	**100**

$P < 0.05$

Table (5): Need of intramuscular pethedine and oral paracetamol among the two groups.

group / analgesic	Caudal bupivacaine	Rectal paracetamol	Total
I. M pethedine	4 (44.4%)	14 (60.8)	18 (56.25%)
Oral paracetamol	5 (55.6%)	9 (39.2%)	14 (43.75%)
Total	**9**	**23**	**32**

P > 0.05

Table (6): Time of administration of intramuscular pethedine and oral paracetamol among two groups.

Group / Time	Caudal bupivacaine	Rectal paracetamol	Total
In first 8 hrs	3 (33.3%)	8 (36%)	11 (34.3%)
After first 8 hrs	6 (66.6%)	15 (64%)	21(65.7%)
Total	**9**	**23**	**32**

$P < 0.05$

Table (7): Complications of caudal bupivacaine and rectal paracetamol among two groups.

Group / Duration	Caudal bupivacaine	Rectal paracetamol	Total
Pain at site of injection	8 (16%)	0	8
No complication	42 (84%)	50	92
Total	**50**	**50**	**100**

$P < 0.05$

Fig. 1: Types of surgery among patients receiving caudal bupivacaine and rectal

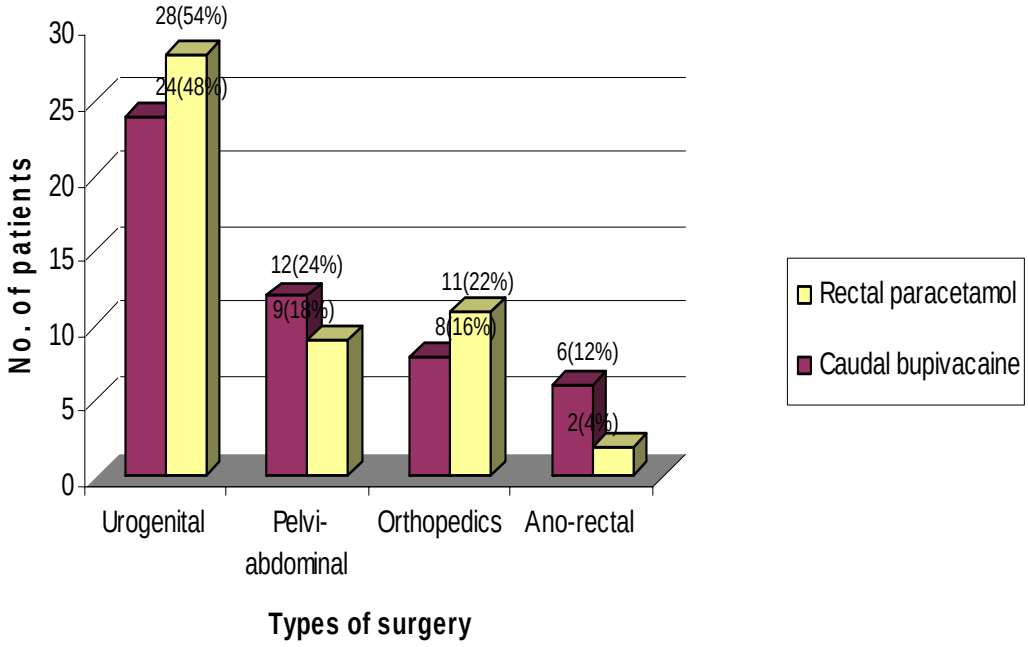


Fig. 2: Changes in postoperative systolic blood pressure among the two groups in the study

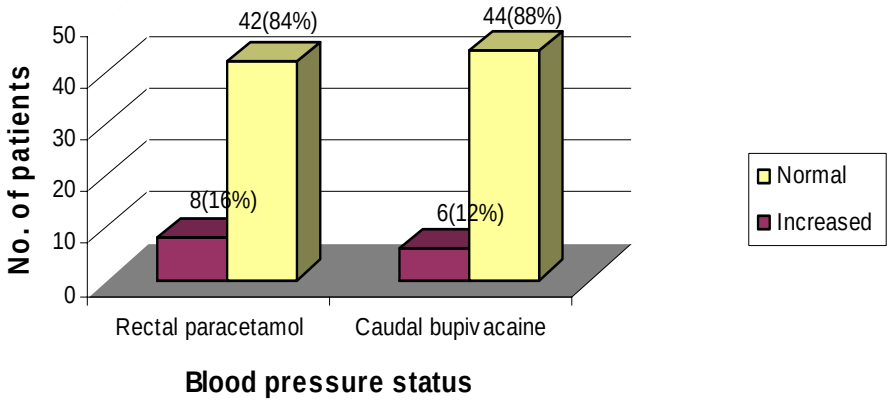


Fig. 3: PDES score among the two groups

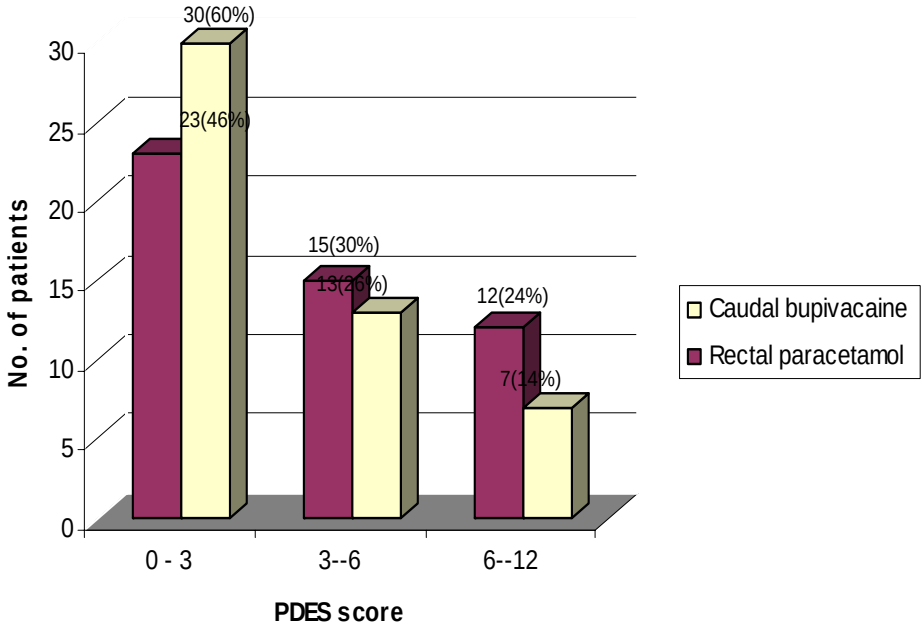


Fig. 4: PDES score among patients undergoing urogenital surgery and other types of surgery receiving caudal bupivacaine

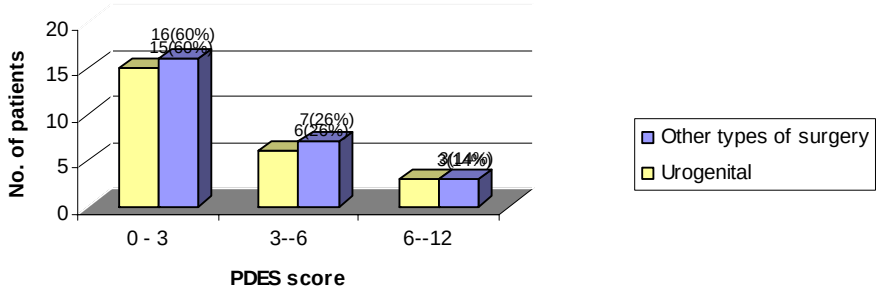
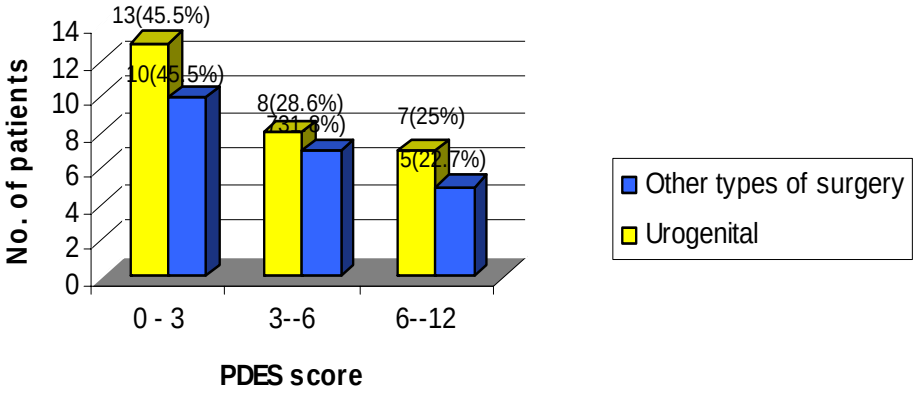


Fig.5: PDES score among patients undergoing urogenital surgery and other types of surgery receiving rectal paracetamol



DISCUSSION

This study compared the postoperative analgesic effect of caudal bupivacaine and rectal paracetamal.

Pain assessment, duration of analgesia and complications were the aspects studied.

The age of patients range from 1- 12 years.

Regional analgesia is being increasingly integrated in the strategy of pain management covering the pre, intra and post-operative period specially in paediatrics

Regional anaesthesia has an established role in paediatrics anaesthesia. New development were increase it's role incoming years for intra and post-operative management.

Peripheral nerve blocks remain under used inspite of their advantages both in term of safety and efficacy.

In this study we demonstrated caudal bupivacaine (0.5 ml/ Kg 0.25 %) produce adequate analgesia in infra-umblical surgery in paediatrics. To calculate the necessary volume and dosages of the anaesthetic we used the formula 0.5 ml /kg.

For rectal paracetamol we used the formula 20 mg/kg.

In our study, the dose and volume of anaesthetic was enough to provide adequate analgesia without complications. All

doses utilize were below that might lead to dangerous plasma level.

The changes in systolic and diastolic blood pressure were minimal. This because the vascular tone is less in children than adults[24]. Heart rate was kept at normal. Regional anaesthesia eliminate the bradycardia response to mesenteric or spermatic cord manipulation during uro-genital or lower abdominal surgery.

Age, weight and sex are not limiting factors for the administration of the techniques[25]. We observed that the younger the patient the more effective the analgesia is.

The opinion of the paediatric surgeons about the regional analgesic technique varied between good and excellent. Caudal bupivacaine has the advantage that the block is more effective within the first 8 hours. (**Table 6**)

The majority of studies concerning postoperative analgesia in pediatrics showed that these drugs provide effective analgesia in pediatrics in different surgical procedures.[18,19,2021] Caudal bupivacaine reduced postoperative opioid consumption.

Eighteen percent of patients who received caudal bupivacaine needed systemic analgesia in the first 24 hrs, 44.4% of them received 1.M pethedine, while 55.6% received oral paracetamol (**Table 4**).

33.3% of patients received analgesic within the first 8 hours, while 66.6% needed analgesia after 8 hours.

46% of the patients who received rectal paracetamol needed systemic analgesic 60.8% of them received I.M pethedine. 66% within the first 8 hours, 39.2% received oral paracetamol. 36% of them within first 8 hours. 46% received analgesic after first 8 hours.

The study by ECK and Roos showed that caudal block is most commonly performed in paediatrics for providing postoperative pain control. The need for postoperative analgesia is only 8.4%. They gave intramuscular morphine.[18]

Another study done by Nielsen and Steels should that regional analgesia in children provides a continuum of perioperative care that include perioperative pain management, decreased opioid requirements, decreased post-operative nausea and vomiting. In addition regional analgesia has been shown to improve the cardiovascular, pulmonary, gastro-intestinal, coagulative, immunological and cognitive functions. And to be of benefit of economic context. [19]

A study done by Wucl and Caldwell revealed that the pathophysiology that commonly followed surgery result in

detrimental physiological effects and may be associated with post-operative morbidity and mortality. The use of epidural analgesia but not systemic opioid may attenuate these effects and facilitate return of gastro-intestinal function, attenuate hyper-coagulable events and decrease post-operative pulmonary complications. And also facilitate patients recovery. [20]

In a double blind study done by Batra, Prasad, Arya, Chari and Yaddanapudi comparing caudal marcaine and tramadol in post-operative pain score and side effects. The result point towards a significantly lower pain score with marcaine and also vomiting is less frequent. [21]

A study of Deing, Diouf and Diene showed that caudal marcaine is safe and secure procedure. Give pain relief even in painful procedures and good post-operative status with only some minor complications. [22]

A study done by Seymour showed that rectal paracetamol is an effective analgesic for controlling post-operative pain. [23]

The only complication encountered by patients who received caudal bupivacaine is pain at the site of injection felt by 16% in this study. In study of Nielsen and steels it was encountered in 4% only.[19]

In this study, caudal bupivacaine proved to be more effective in postoperative pain relief in children than rectal paracetamol but it was associated with a significant incidence of pain at the site of injection.

CONCLUSION

1. Caudal bupivacaine is significantly **m**ore effective than rectal paracetamol in postoperative analgesia in pediatrics, it reduces analgesic requirements.
2. Rectal paracetamol is associated with less complications in comparison with caudal bupivacaine.
3. The time of administration of the conventional analgesic given post-operatively showed no significant difference between caudal bupivacaine and rectal paracetamol.

RECOMMENDATIONS

1. Postoperative analgesic techniques done intra-operatively reduce postoperative pain and it's adverse effects and should be practiced more widely.
2. Caudal bupivacaine is more effective than rectal paracetamol for postoperative analgesia, so it is highly recommended.
3. Further studies regarding the dose and time of administrations should be carried out.

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Table show Pain and Discomfort Evaluation Score (PDES)

Parameters	Criteria	Score
Blood pressure up to	20% of preoperative values	0
	20 - 30% of preoperative values	1
	> 30% of preoperative values	2
Weeping	Without weeping	0
	Weeping, responding to kindness	1
	Weeping, not responding to kindness	2
Movement	None	0
	Unquiet	1
	Agitated	2
Anxiety	Sleeping or calm	0
	Moderate	1
	Very anxious	2
Posture	Without specific posture	0
	Flexion of legs and thighs	1
	Holding the operated site	2
Pain complaint	No pain	0
	Non-localized pain	1
	Localized pain	2

Questionnaire

Postoperative analgesia in pediatrics patients undergoing infra-umbilical surgery: a comparative study between Caudal Marcaine and the Rectal Paracetamol

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- Case No**.....**
- **Patient's Name:** …….....……
- **Weight:** …….....kg
- **Operation:** ……...… •**Duration of surgery:** ……...……
- **Type of analgesia:**

  - Caudal Marcaine ☐ - Rectal paracetamol ☐
- **Time of administration** ……
- **Assessment of pain** …….....……

PDES Scores

Time / min	Score	Time / min	Score
15		120	
30			
45			
60			

- **Analgesic need during the 1st 24 hours:**

  - Yes ☐ - N☐
- **If "Yes":**

  - Drug: ..... **Route:** ..... **Time:**.....